## VIRTUAL HISTOLOGY in AMI

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Clinical Progression of Incidental, Asymptomatic Lesions Discovered During Culprit Vessel Coronary Intervention

NHLBI Registry. 3747 pts 1997-99. 1-yr f/u 216 pts (5.8%) non-target PCI, mean f/u 163  $\pm$  99 days

★86.9% of lesions requiring repeat PCI ≤ 60% at original study

Lesions < 50% at initial angio</p>

Lesions > 70% at initial angio

MVD at original angio

60.5%13.4%76.4%

R. Glaser et al. Circ 2005;111:143

Clinical Progression of Incidental, Asymptomatic Lesions Discovered During Culprit Vessel Coronary Intervention

**Progression in same artery** 39% Progression in different artery 61% **Clinical Presentation: Acute Coronary Syndrome** 68.5% **Stable Angina** 24.1% **Atypical Symptoms** 6.8%

R. Glaser et al. Circ 2005;111:143

Angiographic Prediction of Vulnerable Plaque Is Not Accurate

## **PROSPECT Study**

PI, Dr. Gregg Stone, Guidant, Volcano 3-vessel imaging post PCI of culprit lesion IVUS Virtual Histology F/U: 1 mo, 6 mo, 1 yr, 2 yr, ± 3-5 yr (event driven)

Meds recommended: Aspirin, Plavix 1yr, Statin Repeat imaging in pts with events

Patients enrolled as of 4/5/06 = 639 (from MAHI = 38)



## What is Virtual Histology?

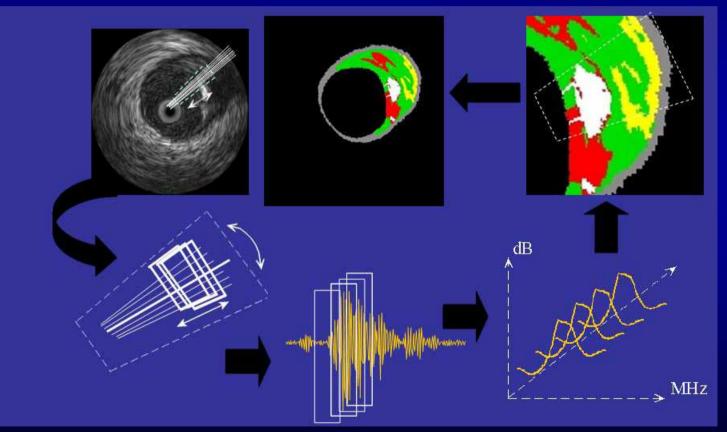
Real-time plaque characterization and visualization with spectral analysis of intravascular ultrasound data

Spectral analysis of the backscattered radiofrequency ultrasound signals allows detailed assessment of plaque composition

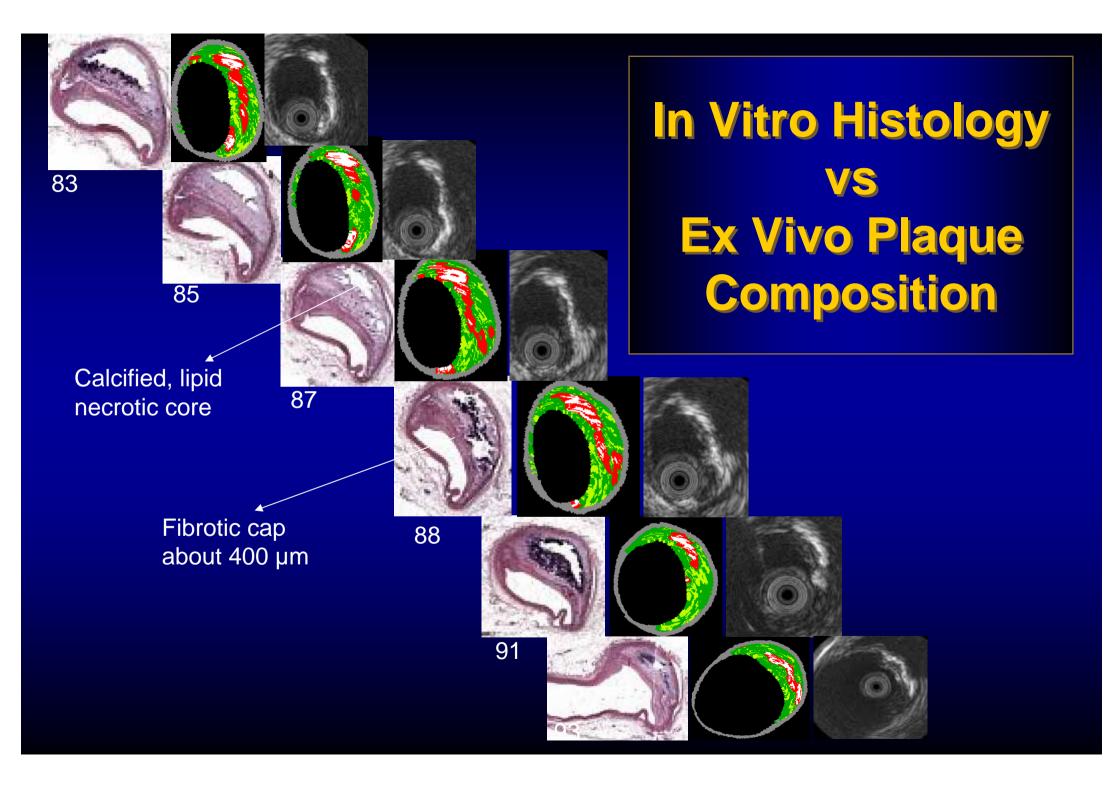
Anuja Nair, Jon D Klingensmith, D Geoffrey Vince, Cleveland Clinic Foundation

## **Image Interpretation**

The Volcano IVUS Program
 In-vivo characterization of plaque composition via advanced spectral analysis



#### Fibrous; Fibro-lipidic; Lipidic-necrotic; Calcium

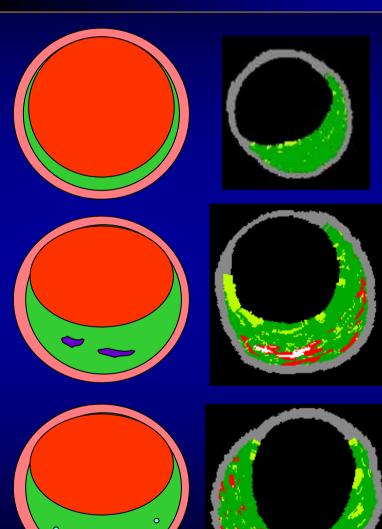


### EAGLE EYE JUNE 2005 CLASSIFICATION TREE Accuracy Data:

Slice by Slice VH & Histology Comparison
Overall accuracy for ROIs – 94.96%
\$51 LADs, 115 Artery Sites, 407 Total ROIs

	Sensitivity	Specificity	Predictive Accuracy
	(%)	(%)	(%)
Fibrous Tissue ( <i>n</i> = 162)	83.95	98.78	92.87
Fibro Fatty ( <i>n</i> = 84)	86.90	95.05	93.37
Necrotic Core ( <i>n</i> = 69)	97.10	93.79	94.35
Dense Calcium ( <i>n</i> = 92)	97.83	99.68	99.26

#### **Generally Stable Plaque Types\***



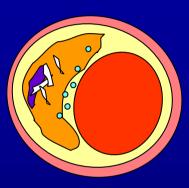
**<u>"Fibrous"</u>** – Plaque nearly all fibrous tissue.

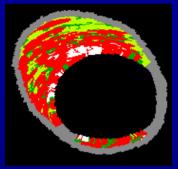
<u>"Fibro-Calcific"</u> – Mainly fibrous with some Dense Calcium. Necrotic Core 3-10%

"Pathological Intimal Thickening" –Fibro-Fatty and Fibrous tissue, with Necrotic Core 0-3% due to micro-calcifications within the Fibro-Fatty tissue. Possible progression to risky atheroma.

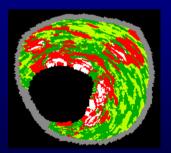
\*Courtesy of Renu Virmani

## Thin-Cap FibroAtheroma (TCFA)

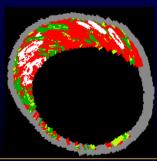




"Thin Cap Fibro-Atheroma (TCFA)" or "Vulnerable Plaque" - Necrotic Core >10% of total plaque volume and located at or near the lumen.



<u>"TCFA with significant narrowing</u>" ( $\geq$ 50% reduction in Cross Sectional Area on IVUS or DS $\geq$ 25% on angiogram)

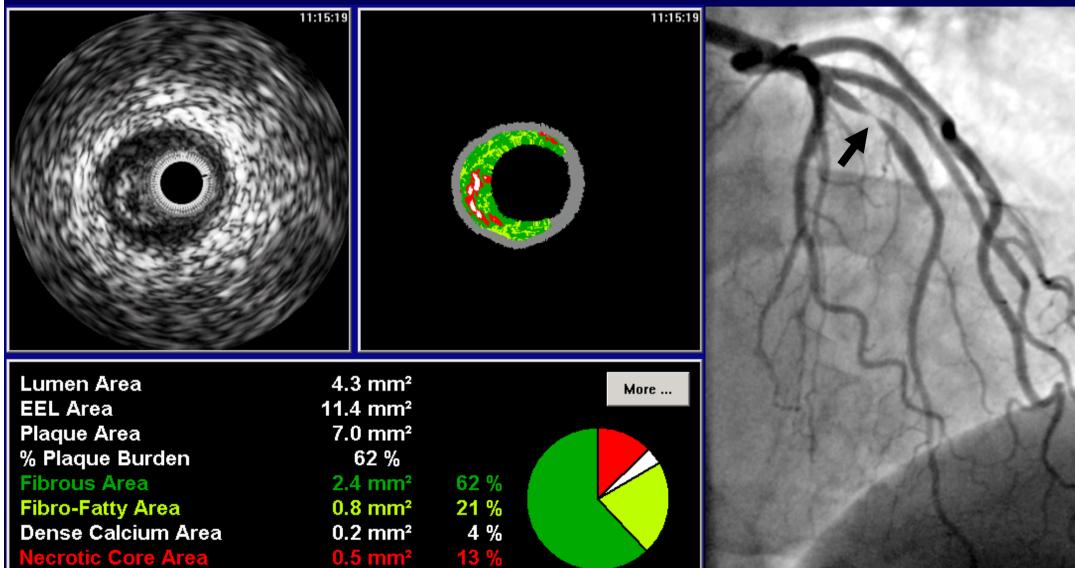


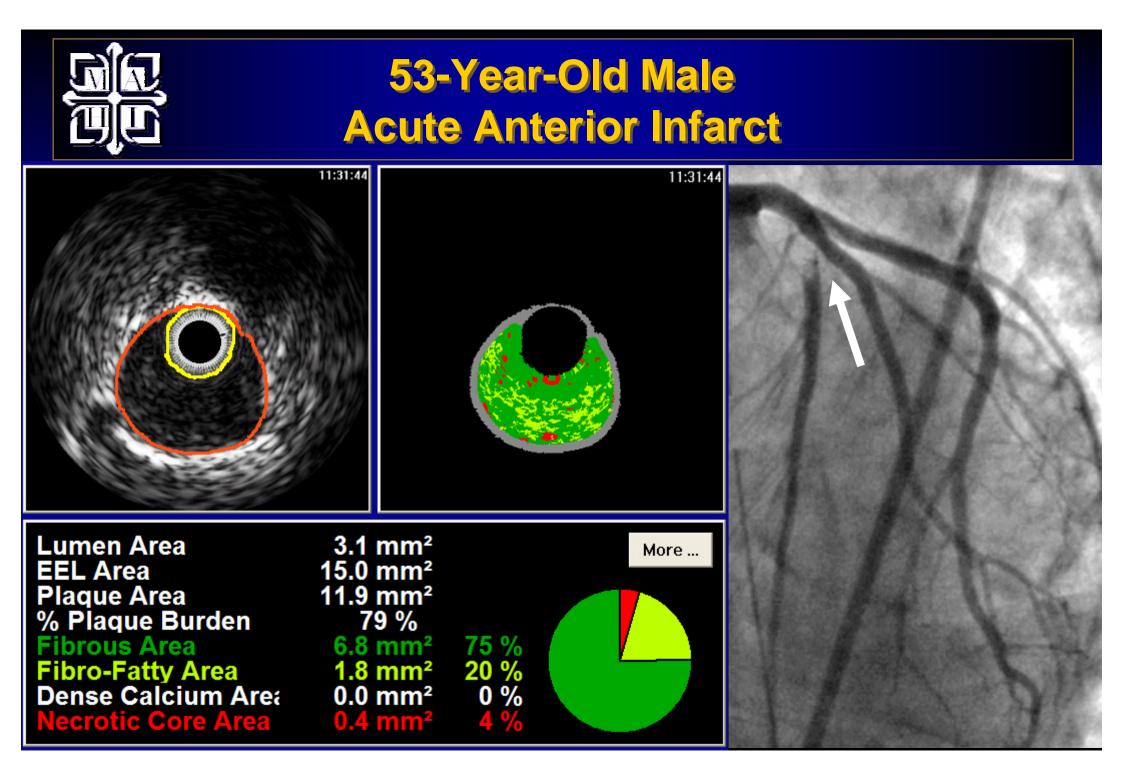
<u>"TCFA without significant narrowing"</u> (<50% area reduction on IVUS or <25% DS on angiogram)

**Courtesy of Renu Virmani** 



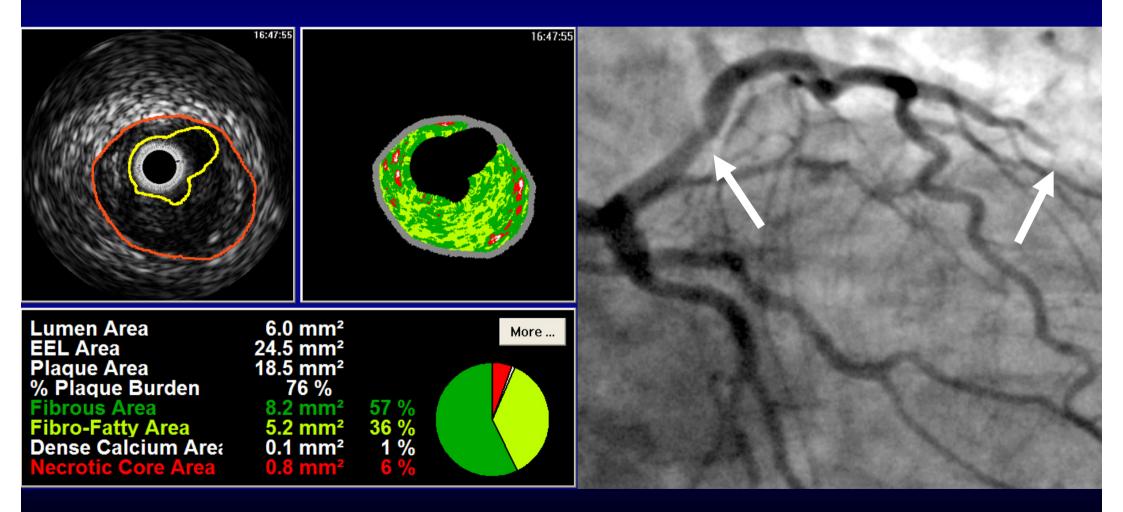
#### **53-Year-Old Male** Acute Non-STEMI





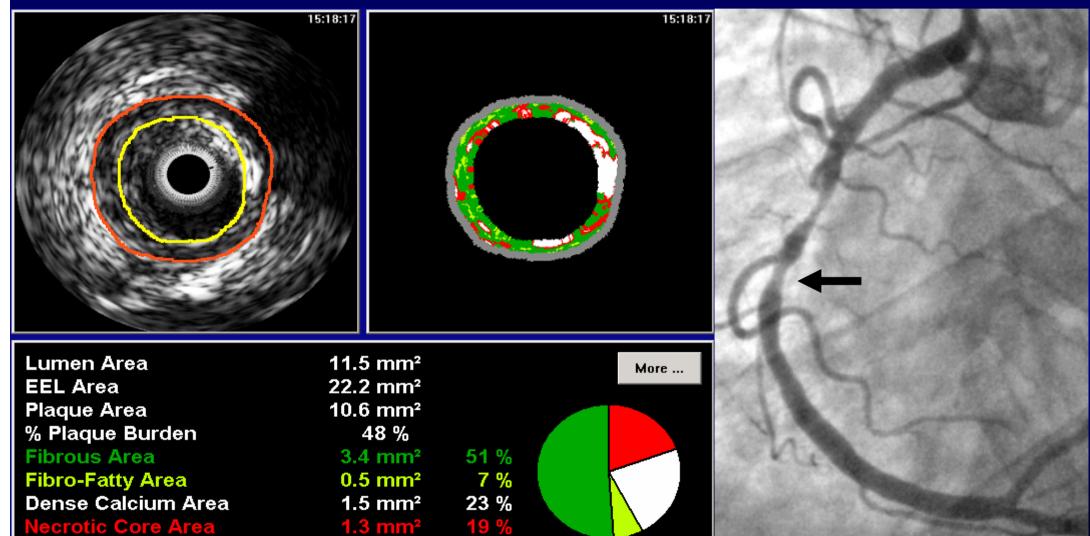


#### 58-Year-Old Male Acute Lateral Wall Infarct



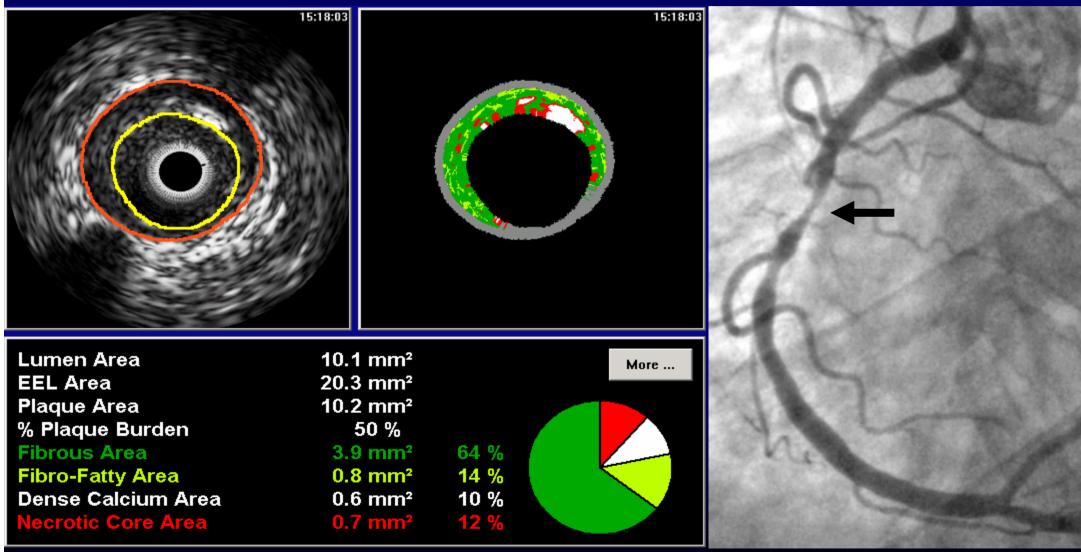


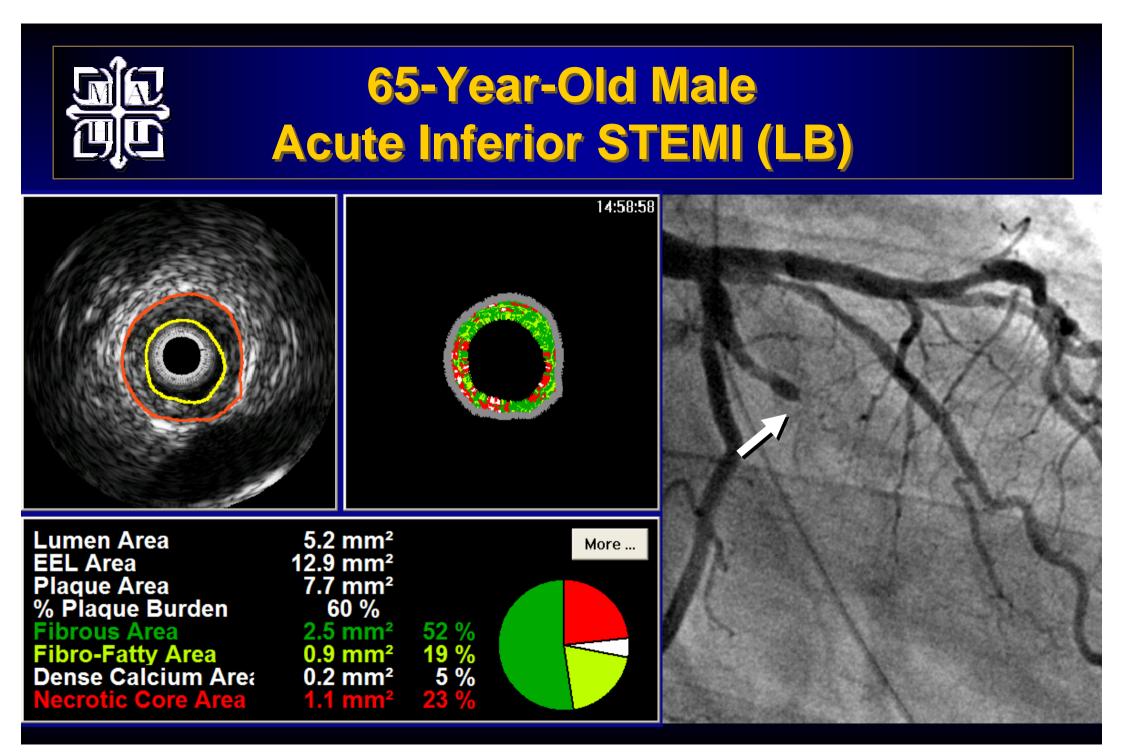
## 52-Year-Old Male Acute Inferior Infarct

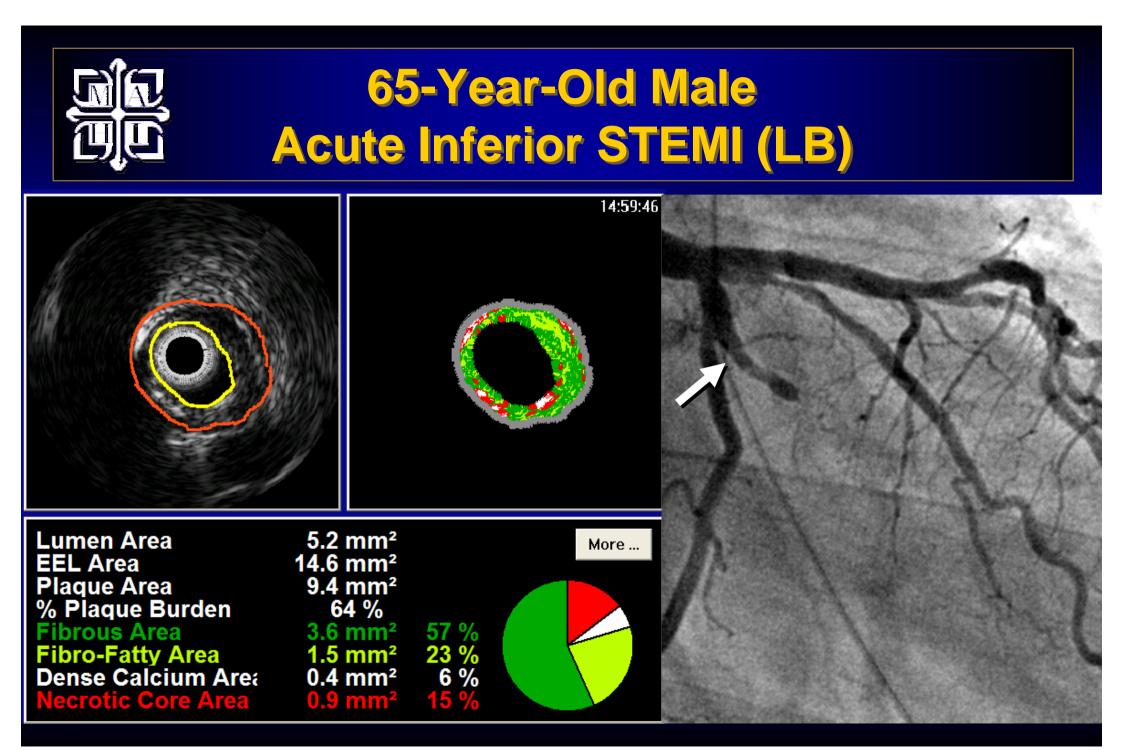


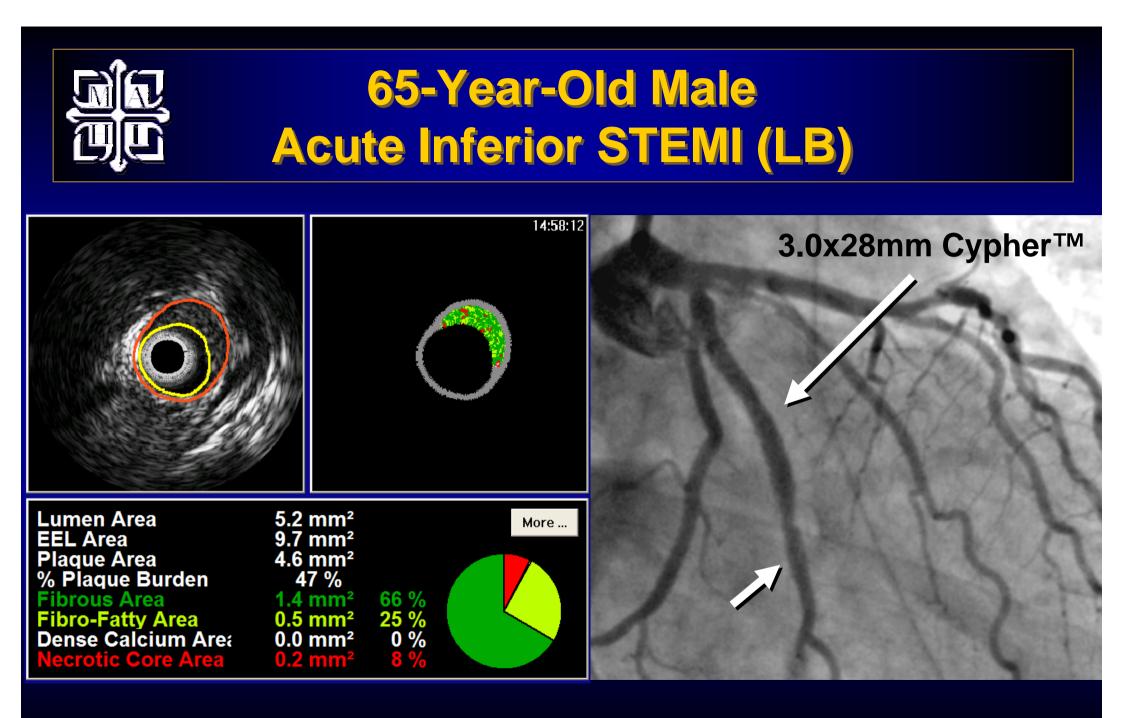


### 52-Year-Old Male Acute Inferior Infarct





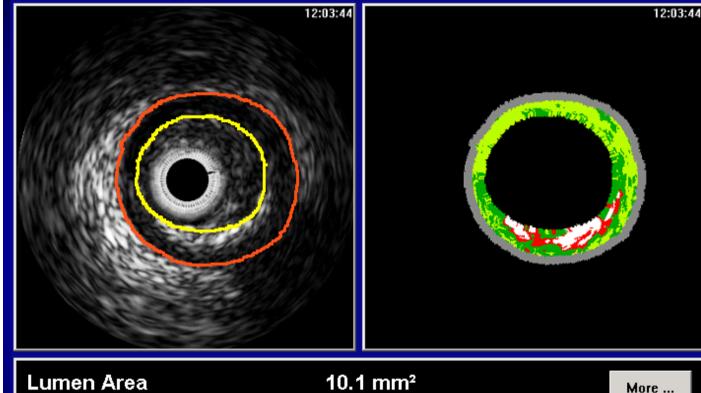




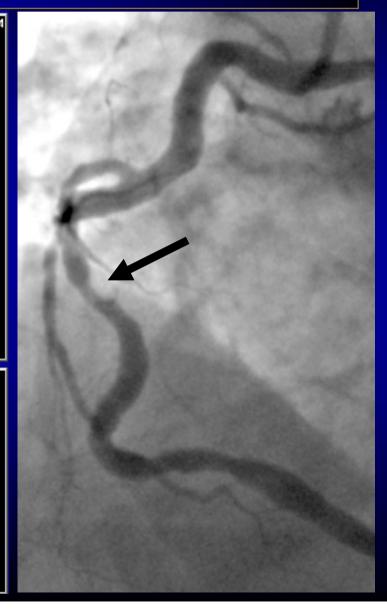


Lumen Area	8.9 mm <sup>2</sup>		More	Carlos Andrews
EEL Area	17.6 mm <sup>2</sup>			
Plaque Area	8.7 mm <sup>2</sup>			
0/ Blogue Burden	50 %			
% Plaque Burden		0.0 1/		
Fibrous Area	3.2 mm <sup>2</sup>	63 %		
Fibrous Area Fibro-Fatty Area	3.2 mm <sup>2</sup> 1.1 mm <sup>2</sup>	21 %		
Fibrous Area				

#### 63-Year-Old Male Acute Inferior Wall Infarct - RCA



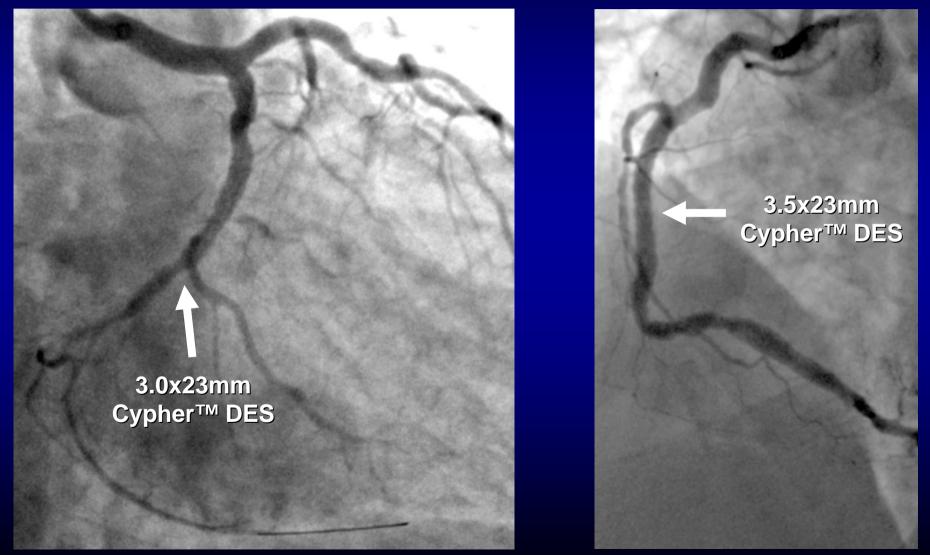
Lumen Area 10.1 mm<sup>2</sup> EEL Area 21.7 mm<sup>2</sup> Plaque Area 11.6 mm<sup>2</sup> % Plaque Burden 54 % Fibrous Area 2.8 mm<sup>2</sup> 36 % Fibro-Fatty Area 3.4 mm<sup>2</sup> 44 % Dense Calcium Area 0.8 mm<sup>2</sup> 11 % Necrotic Core Area 0.7 mm<sup>2</sup> 9 %

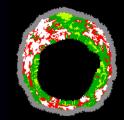


63-Year-Old Male Acute Inferior Wall Infarct - LAD							
	11:55:43						
Lumen Area EEL Area	9.5 mm² 20.9 mm²	More					
Plaque Area	11.5 mm <sup>2</sup>						
% Plaque Burden	55 %						
Fibrous Area	3.9 mm <sup>2</sup>	52 %					
Fibro-Fatty Area	2.0 mm <sup>2</sup>	27 %					
Dense Calcium Area	0.6 mm²	8 %					
Necrotic Core Area	1.0 mm <sup>2</sup>	13 %					



#### 63-Year-Old Male Acute Inferior Wall Infarct





## Virtual Histology in AMI

 Good characterization and visualization of plaque composition & volume (plaque burden)
 Careful border recognition to exclude thrombus
 Surprisingly widespread distribution of vulnerable plaques in pts with ACS – relating to both culprit and non-culprit arteries

"Systemic Vascular Illness"

# IVUS-VH Derived Plaque Phenotyping IVUS-VH Derived Plaque Phenotyping

IVUS-VH imaging in 76 pts prior to PCI 209 lesions of interest evaluated

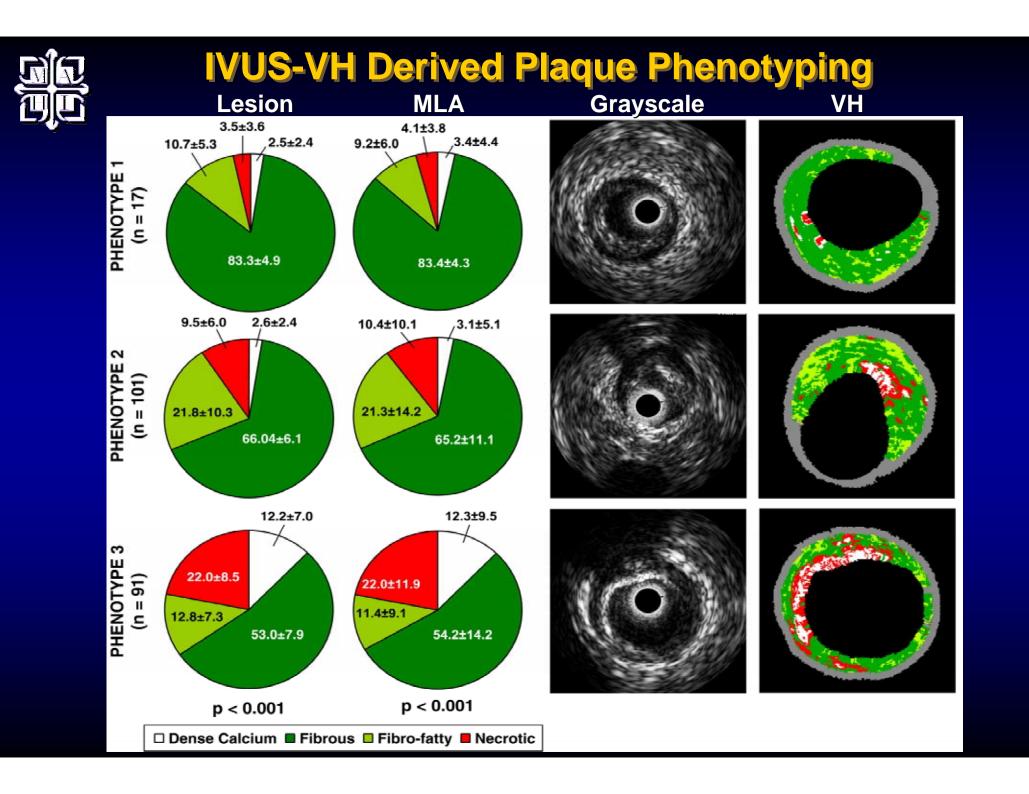
20 MHz catheter (Eagle Eye, Volcano Corp, CA) Automated pullback 0.5 mm/second

Utilizing a latent class cluster technique, 3 unique phenotypes were identified



### **IVUS-VH Derived Plaque Phenotyping**

	Phenotype 1 (n=17)	Phenotype 2 (n=101)	Phenotype 3 (n=91)	P-value
Segment length (mm)	16.79 ± 12.33	15.06 ± 9.62	17.51 ± 10.55	0.03
Seg vol plaque burden (mm <sup>3</sup> )	43.66 ± 6.91	46.41 ± 11.53	50.93 ± 10.31	0.003
MLA plaque burden (%)	52.58 ± 9.82	55.96 ± 14.75	61.48 ± 12.38	0.004
Stenosis at MLA (%)	24.93 ± 19.59	31.28 ± 18.27	37.52 ± 16.97	0.007
MLA vessel EEL CSA (mm <sup>2</sup> )	11.13 ± 5.27	15.42 ± 5.85	14.33 ± 4.91	0.009
MLA vessel min diam (mm)	3.42 ± 0.83	4.03 ± 0.80	3.88 ± 0.70	0.009
MLA lumen CSA (mm <sup>2</sup> )	5.09 ± 2.23	6.64 ± 3.55	5.35 ± 2.30	0.006
MLA plaque CSA (mm <sup>2</sup> )	6.03 ± 3.43	8.78 ± 4.43	8.98 ± 4.05	0.03
Remodeling index at MLA	0.96 ± 0.17	0.94 ± 0.15	0.92 ± 0.16	0.6
Eccentricity	0.43 ± 0.20	0.58 ± 0.19	0.58 ± 0.17	0.06





## **IVUS-VH Derived Plaque Phenotyping**

#### **Conclusions:**

- IVUS-VH is a feasible imaging platform to define lesions in target vessels of PCI patients
- There were 1.5 non-culprit lesions for every 1 culprit lesion in target vessels
- Identified 3 distinct phenotypes that differed with respect to composition and plaque burden (p < 0.001)</p>
- Tight correlation between lesion phenotype and mean lumen area plaque composition (p < 0.001)</p>



#### **Conclusions:**

**Among pts with > one lesion:** 

- -34 (46%) had lesions of single phenotype
- -37 (50%) had lesions of two phenotypes
- -3 (4%) had lesions of three phenotypes
- Trend for clustering of culprit plaques to phenotypes 2 and 3 (p = 0.077)